Treatment of acute viral croup

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SUMMARY Total respiratory resistance (R_T) was measured before and after nebulised α -adrenergic stimulant therapy in 8 children aged 4 to 18 months who had the clinical symptoms of acute viral croup. In 7 children there was a mean fall in R_T of 30% after treatment, associated with an improvement in their clinical condition. This improvement was shortlived, the resistance returning to pretreatment levels within 30 minutes. The remaining child showed no improvement after phenylephrine but was subsequently found to have acute epiglottitis. Nebulised water did not produce any change, indicating that the response was not due to moisture alone.

Acute viral croup is a common respiratory illness in infants and young children. Most patients recover spontaneously at home but of those admitted to hospital, 1-2% will require endotracheal intubation. There is much controversy regarding the use of treatment with cold water mist in croup but no evidence that this is of any benefit. Indeed, there is no evidence that any drug effectively alters the natural history of an attack of croup. Nebulised racemic epinephrine (adrenaline hydrochloride) administered either by a compressor and face mask or by intermittent positive pressure breathing (IPPB) has recently become widely used in North America. There have been several uncontrolled studies advocating its use but in a controlled study Gardner et al. (1973) were unable to show any difference between a group of children receiving racemic epinephrine and a group receiving sterile saline placebo. They suggested the apparent effectiveness of racemic epinephrine might be due to the nebulisation of moisture rather than a direct effect of the drug. Taussig et al. (1975) however showed an immediate improvement in clinical signs and symptoms after administering racemic epinephrine by IPPB but the symptoms returned within 2 hours, there was no improvement in arterial oxygen tension, and the duration of the illness was unchanged compared with a control group.

Epinephrine has both α - and β -adrenergic stimulant properties. It is well known that α -adrenergic stimulants, such as xylometazoline, reduce infective nasal mucosal oedema and we considered it likely that it was the α - rather than the β -adrenergic stimulant property of racemic

epinephrine which improved the clinical state of children with croup.

To our knowledge no attempt has been made to measure objectively lung function changes after treatment in croup and there is much confusion regarding the usefulness of moisture and adrenergic therapy in the illness. The aim of this study was to evaluate the usefulness of both nebulised cold water and a nebulised α -adrenergic stimulant drug in acute viral croup by comparing total respiratory resistance (R_T) measurements before and after treatment. The α -adrenergic stimulant we chose was phenylephrine.

Subjects

Eight boys were studied as inpatients, their ages ranged from 4 to 18 months (mean 9 months). Apart from the attack of croup they were well. One baby had experienced an attack of croup previously. There was no family history of respiratory illness or atopy in first-degree relatives. The diagnosis of croup was made on history and clinical examination. Viral studies were not performed in any child. At the time of testing all babies were in the recovery phase of the illness but still had mild respiratory stridor at rest. Permission for testing was obtained from at least one parent and the mother of each child was present during the test. The study had been passed in advance by the Nottingham Ethical Committee.

Method

Total respiratory resistance $(R_T)^T$ was measured using a modification of the forced oscillation technique (Cogswell, 1973). The apparatus and

technique have been described (Lenney and Milner, 1978a, b). R_T was measured at midinspiration throughout this study as the stridor most affected this part of the breathing cycle.

To ensure fully patent nasal passages 2 drops of xylometazoline (0.05% solution) were instilled into each nostril. Each child was then sedated with 80 mg/kg chloral hydrate. When asleep the child was placed supine on a couch and the face mask slowly lowered over the mouth and nose using gentle pressure until an airtight seal was obtained. Traces were obtained from five or six breaths. R_T was expressed as the mean of at least 6 readings. R_T measurements were repeated 10 minutes later. Five of the children were then given 2 ml sterile water using a Pari nebuliser and R_T measurements were repeated 5, 10, 15, and 25 minutes later. These 5 children and the remaining 3 children were then given 2 ml 0.25% phenylephrine solution using the Pari nebuliser and the measurements repeated 2, 5, 10, 15, 20, 25, and 30 minutes later. Three children awoke approximately 30 minutes after the phenylephrine inhalation but in the remaining 5 children a further 2 ml 0.25% phenylephrine solution was given using the Pari nebuliser and R_T measurements were repeated 2, 5, 10, 15, 20, 25, and 30 minutes later.

Pulse rate was measured before and after each inhalation of water and phenylephrine. Blood pressure was measured before and after each phenylephrine inhalation using the Doppler technique.

Results

All children had initial $R_{\rm T}$ values much higher than expected from extrapolation of available data (Cogswell, 1973). They ranged from 5·2 to 7·8 kPa/l per s (52 to 78 cmH $_2$ O/l per s) whereas the expected range was 1 to 3·5 kPa/l per s (10 to 35 cmH $_2$ O/l per s). $R_{\rm T}$ measurements 10 minutes later were similar to the initial readings, the coefficient of variation between the two readings being 5·9% giving a correlation coefficient (r) of 0·96 (P<0·001).

The 5 children given nebulised water showed no striking change in R_T after the inhalation. The greatest fall in R_T was 7% and the greatest rise 24%. The mean percentage rise in R_T at 5, 10, 15, and 25 minutes after the nebulised water was 7.5, 5.8, 7.6, and 7.2% respectively. There was no clinical change in any of the 5 children after the nebulised water, the inspiratory stridor remaining unchanged.

Seven of the 8 children given nebulised phenylephrine showed a striking fall in R_T after treatment. The mean maximum fall in these 7 children at 15 minutes after therapy was 30% (range 17–38%)

(Figure). The fall in $R_{\rm T}$ was accompanied by a definite reduction in inspiratory stridor in 3 patients and by complete disappearance of the stridor in 4. The improvement was short-lived and as $R_{\rm T}$ increased the stridor returned. 30 minutes after treatment the mean $R_{\rm T}$ level was 6% higher than it had been before.

The 5 children who remained asleep were given a further inhalation of phenylephrine, and in 4 of these children $R_{\rm T}$ fell strikingly. The mean maximum fall in these 4 children was 35% (range 27–46%) and, in all 4 children, the inspiratory stridor disappeared. 30 minutes after the second inhalation, the stridor had fully returned and the mean $R_{\rm T}$ was only 8% lower than before treatment.

One child showed no fall in R_T greater than 7% and no improvement in the stridor after the two inhalations of phenylephrine. At the end of the test his stridor appeared marginally worse and during the next few hours his condition deteriorated. Blood cultures and throat swab grew *Haemophilus influenzae* and he eventually made a full recovery after a course of parenteral ampicillin.

Pulse rate was unchanged in any child after treatment with water or phenylephrine. Blood pressure was unchanged after phenylephrine except in 2 babies in whom the systolic pressure rose from 100 to 110 and from 100 to 115 mmHg after the second inhalation of phenylephrine.

Discussion

Racemic epinephrine administered by a compressor and face mask or by IPPB has been used in the treatment of acute viral croup but doubt has been cast on its value. Even those who consider it to be

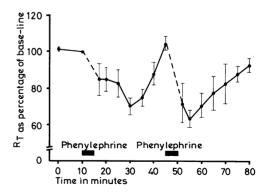


Figure Total respiratory resistance (R_T) before and after two 5-min inhalations of phenylephrine (mean for group \pm 1 SE) in a group of 7 children aged 4 to 18 months with croup. Only 4 of these children received the second inhalation of phenylephrine.

effective find that the therapeutic response is shortlived and its use should be limited to children in hospital (Taussig et al., 1975). In this study we have objectively assessed the response to α-adrenergic stimulant therapy in 8 children aged 4 to 18 months with clinical symptoms of acute viral croup. The results show that 7 of the 8 children who had symptoms of croup at the time of study responded clinically, objectively, and reproducibly to nebulised phenylephrine. The response however was disappointingly short, the resistance returning to pretreatment levels within 30 minutes. None of the 5 children given nebulised water showed any improvement either clinically or objectively during a 30minute period so we do not consider the response to nebulised phenylephrine was due to nebulised moisture alone. This failure to show any improvement in lung function after nebulised water is noteworthy because to our knowledge this is the first time that anybody has tried to assess objectively the response to nebulised water in acute viral croup. It was interesting that the one child who did not improve after nebulised phenylephrine gradually deteriorated and H. influenzae was grown from his blood and throat indicating that he had acute epiglottitis rather than viral croup.

At this stage we would not advocate the use of phenylephrine in croup because the response appears to last for so short a time and in one child who responded to the phenylephrine there was clinical evidence of deterioration on returning to the ward possibly owing to a rebound phenomenon. If this rebound phenomenon does exist it could have serious repercussions at the height of an attack. We do consider the results interesting, however, and suggest that further evaluation of other α -adrenergic stimulant drugs is worthwhile. It may well be that oral α -adrenergic stimulant drugs have a part to play in the management of acute viral croup.

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